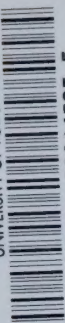


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by

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STUDY NO. 4

TRAUMATOGENIC OCCLUSION AND ARTICULATION



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STUDY NO. 4

TRAUMATOGENIC OCCLUSION AND ARTICULATIONOutline - A. Individual Loading versus Collective Loading

B. Individual Loading: Traumatogenic Function

C. Factors that tend to lead to Trauma

D. Axial and Oblique Loading

E. Axial and Oblique Overloading

F. Comparison of Axial and Oblique Loading in Relation to
Overstress in the Periodontal Membrane

G. Some Causes of Traumatogenic Occlusion

- (a) Certain observations of Neustadt
- (b) Observations of Hanau
- (c) Mild function and traumatogenic occlusion

H. Bibliography

TRAUMATOGENIC OCCLUSION AND ARTICULATION

A. Individual Loading versus Collective Loading

When, under favourable conditions, wear assumes its proper place in the physiology of the human masticatory apparatus, interfering cusps tend to be removed. The associated teeth show a change from relatively individual loading to collective loading.

Two main benefits accrue from this change, viz:

- (a) Decrease in magnitude of the occlusal force per tooth
- (b) Decrease in the angle of contact

A third benefit arises in these cases where there is a change from overlapping in the incisor region, e.g. in the Australian aboriginal.¹ When, in the anterior region the teeth move into an end-to-end occlusion, there is a shift in the teeth axes which tends to be beneficial. These points will be dealt with at greater length later on in this paper.

B. Individual Loading

Ideal coördination of the occlusal inclined planes of the teeth, in centric and the eccentric positions, is extremely rare. Cusp interference, in varying degree, through horizontal malposition of teeth is the rule. When this is allowed to persist, mainly through lack of wear, individual loading in certain positions remains. As a result, the magnitude of the occlusal force is borne by the tooth that is contacted first, and besides, the original angle of contact remains unchanged. Also the normal shifts in the axes of the anterior teeth fail to take place.

There is presented here in contrast to heavy function within the limits of the reserve force of the tissues, a type of function that tends to be injury-producing. To this condition the writer has applied the term "traumatogenic function". (Gr. trauma, a wound; and the root 'gen', to produce)

An occlusion is known as a contact relation of the masticatory surfaces of the teeth. (Hanau)² When an occlusion, under biting pressure, produces an injury in the periodontal tissues, it is known as a "traumatogenic occlusion". As the term articulation expresses the dynamic relationship of the masticatory surfaces during the natural movements of function, it follows that such a relationship when it brings about injury to the supporting tissues of the teeth is a "traumatogenic articulation".

According to the viewpoint of the writer,³ the term "traumatic occlusion" should be employed only when it is meant that the contact relation of the masticatory surfaces of the teeth is directly the result of trauma. There are presented in practice occlusions which are clearly the result of trauma and occlusions which produce trauma. According to our usage of these terms, the meanings conveyed are completely opposite.

4. Individualism

There is a certain amount of individualism in the American mind. It is not a very deep individualism, but it is a certain amount of individualism. It is a certain amount of individualism.

The individualism of the American mind is a certain amount of individualism.

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According to the viewpoint of the individual, the individual is a person who is not a part of a group. He is a person who is not a part of a group. He is a person who is not a part of a group.

C. Factors that Tend to Lead to Trauma

- (a) Excessive magnitude of the occlusal force, e.g. interfering cusps; tooth elevation.
- (b) Excessive angle of contact, e.g. lack of wear; certain examples of excessive wear.
The angle of contact is defined as the angle between the axis of the tooth and the perpendicular to its surface at the point of contact. (Professor Synge)[#]

Immediate Causes of Trauma

- (a) Excess of pressure in the periodontal membrane
- (b) Defect of pressure in the periodontal membrane

Excess of pressure exists when a load is applied to the tooth and the pressure rises considerably above atmospheric in some zones of the periodontal membrane. (The pressure throughout the periodontal membrane of an unloaded tooth is atmospheric, i.e. 15 lb. per sq. in.) When the pressure falls below atmospheric in other zones of the periodontal membrane, there is said to be a defect of pressure.

D. Axial and Oblique Loading

In axial loading on the tooth, the perpendicular to the tooth at the point of contact is along or parallel to the axis of the tooth. According to Hay^{*4} the pressure in the periodontal membrane exceeds atmospheric pressure throughout and is greater at the apex than along the side of the root.

In oblique and transverse loading, in which the force is exerted in a direction oblique or perpendicular to the axis, the zones of excess of pressure are found in the gingival third on the side away from the point of application, and in the opposite apical third. Here, according to the computations of Hay, made on conical roots, the greatest excess of pressure is to be found. The defects of pressure are found in the pull zones, the gingival one being greater than the apical.

When these excesses and defects of pressure are small, they may be regarded as normal and not traumatogenic.

E. Axial and Oblique Overloading

(a) Axial Overloading When an excessive axial load is applied to the tooth, the excess of pressure is greatest at the apex, and in this region it tends to become traumatogenic. Obviously the tissue alterations in the periodontal membrane occur mainly in the periapical zone, the radiograph being the logical means of their detection.⁵

J. L. Synge, Professor of Applied Mathematics, University of Toronto.

* The investigations of Mr. G. E. Hay, M.A., were conducted with the aid of the National Research Council of Canada.

(b) Oblique Overloading In the case of excessive oblique or transverse loading, the regions in the periodontal membrane subject to trauma are those of excess of pressure and defect of pressure. Thus we may expect to find in the regions of excess of pressure characteristic lesions such as necrosis, collagenous change etc., and in regions of defect of pressure lesions such as cemental fracture.

F. Comparison of Axial and Oblique Loading in Relation to Overstress in the Periodontal Membrane

Let us consider a load of 3 oz. applied (a) axially, and (b) obliquely at an angle of 45° to the axis of the tooth. According to the calculations of Hay, the greatest pressure in case (a) occurs at the apex and is approximately 22 lb. per square inch. (This is an excess of 7 lb. per square inch over atmospheric.) On the other hand, in case (b), the greatest pressure occurs in the apical third on the side of the application of the force and is approximately 30 lb. per square inch. If we assume that the limit of safety for the tissues is approximately 25 lb. per square inch, we see that in the case of (a) the pressure at the apex is within the normal physiological limits, while in the case of (b) the load is traumatogenic. This is also true for the gingival third on the side away from the load where the pressure in the periodontal membrane is approximately 27 lb. per square inch.

The difference between (a) and (b) arises solely from a difference in the angle of contact, the magnitude of the occlusal force being the same in each case. On this basis, therefore, the definition or conception that traumatogenic occlusion is an excessive occlusal force is not fundamentally correct. While excess of magnitude of the occlusal force is undoubtedly the greatest single factor in the production of traumatogenic occlusion, it can be seen from the foregoing demonstration that the angle of contact is of extreme importance also. The essence of traumatogenic occlusion however produced--the thing that constitutes its particular nature and makes it what it is--is overstress in the periodontal membrane. And, in the majority of severe forms, excessive magnitude of the occlusal force is combined with excessive angle of contact.

It will be of interest at this point to refer to contact relationships in the incisor region known as "end-to-end" and "overlapping" occlusions. According to Synge, an end-to-end occlusion in the incisor region may be defined as a contact relationship in which the axes of the teeth intersect the area of contact.

An end-to-end occlusion benefits the lower tooth by establishing an axial loading, and the upper tooth by changing the angle of contact arising out of the shift in the axis. As pointed out previously, this change takes place in well-worn dentitions, and is common in primitive peoples.

An "overlapping" occlusion in the incisor region is usually regarded as a type of vertical overbite, and obviously both axes of the teeth do not intersect the area of contact.

G. Some Causes of Traumatogenic Occlusion

(a) Certain Observations of Neustadt⁶

Neustadt in his classic treatise, "Normal and Abnormal Occlusal Relations", has pointed out that in an approach to the subject of traumatogenic occlusion two things are necessary, viz, a complete understanding of the constitution of normal occlusion, and of the different ways the dental arch may be affected by abnormalities of alignment. The writings of this investigator have greatly served to illuminate the problem of occlusal relationships, and their careful study is recommended to all students of periodontology.

Neustadt has thoroughly described the clinical occlusal picture when coordination is present in centric as well as the eccentric positions and has called attention to the fact that this ideal normal is seldom encountered in the mouth. Minor deviations are cared for by such agencies as natural wear, continuous tooth elevation, and tooth depression. He believes that the commonest cause of occlusal incoordination is brought about by malpositions of the teeth. These include the horizontal malpositions, viz, mesio-distal, labio-lingual, and the rotations, and they cause traumatogenic occlusion "because they take the cusps of the teeth out of their normal occlusal relationship". Examples are seen where a cusp bites on the marginal ridge instead of the normal groove. Cusp interference, then, results from slight horizontal malpositions. When the malpositions are more extreme, a change in the vertical position of the tooth is involved.

According to Neustadt, traumatogenic occlusion is caused either by cusp interference or excessive elevation. In an earlier paper,⁷ he differentiated between tooth eruption and tooth elevation, pointing out that "in the common use of the term, 'tooth eruption', two phenomena have been included which, in reality, are of an entirely different nature." Tooth eruption refers "solely to the length of the tooth portion visible in the mouth from occlusal surface to gingival attachment, regardless of the relation of the tooth to the plane of occlusion." Occlusal tooth elevation refers to "the position of the tooth regarding the plane of occlusion, irrespective of the relationship of the gingiva to the tooth." The tooth and its supporting tissues are carried toward the plane of occlusion en masse. This tendency to elevate belongs to every tooth, and the elevation is operative until certain resistance is encountered. This is normally furnished by the opposing tooth. Then, the elevating force is inhibited, becoming operative again on removal of the occlusal impediment. It has been pointed out by Neustadt that, in the posterior teeth, the production of excessive elevation and consequent traumatogenic occlusion is largely caused by extractions. (The untimely loss of deciduous teeth plays an early part in excessive elevation. The relationship between their early loss and undue retention, through a disturbance of harmony of eruption of the permanent molars, frequently has a bearing on the production, on both sides of the mouth, of dissimilar states of occlusal elevation of the bicuspid. Later, the loss of molars and bicuspid often results in excessive elevation of the antagonistic teeth. Malpositions of the posterior teeth as related to excessive elevation, are of secondary importance in the establishment of traumatogenic occlusion. These teeth are wide labio-lingually thus providing a fully sufficient occlusal contact.

Horizontal malpositions of the incisor teeth, on the other hand, are the commonest cause of traumatogenic occlusion in the anterior part of the mouth. The incisors are narrow labio-lingually, and only slight horizontal shifts in position are necessary as a rule to free the teeth from contact. This will allow both the upper and lower teeth involved to elevate without restraint with the end result of vertical malposition. It is pointed out also, that the anterior teeth are very subject to malpositions due to the influence on them of a number of factors including habits of lip and tongue, and drifting caused by loss of teeth in the posterior region.

(b) Observations of Hanau (as related to the writer)

Disuse and the Production of Traumatogenic Occlusion

The Matabeles coming from their native kraals to work their indentureships in the mines, leaving behind their coarse native diet, are forced, more or less suddenly, to adapt themselves to a soft European diet. After some years (3-5) these natives return to their original environment and diet. Hanau found, following these events, in general and varying in degree, a progressive loss of the teeth from the third molars forward. He explained this phenomenon as follows: Beginning with a "rocking chair" occlusion, which implies a lack of contact of the second and third molars in centric occlusion and a splendid coordination in the lateral positions and movements, through relative disuse a marked change apparently took place in these dentitions during the interval at the mines. Having little need to use the lateral articulations, on account of the softness of the food, through eruption or tooth elevation, the second and third molars moved into occlusal contact in centric occlusion. An up-and-down movement with centric contacting of the occlusal surfaces, sufficed in mastication. Returning to their native diet in the kraals, where stronger and more extensive functioning movements of the mandible are necessary to comminute the food, these teeth, viz: the second and third molars, are found to be in traumatogenic occlusion, and their loss followed in due time.

(c) Mild Function and Traumatogenic Occlusion

Lower molars occasionally are found in a position to the lingual of the upper molars. They elevate into supra-occlusion and, not contacting in centric occlusion, are in reality mildly functioning teeth. Yet in lateral movements of the mandible, they frequently come into a severe form of traumatogenic occlusion. This also is often the case in the upper anterior region, e.g. the lateral incisor. The periodontal membrane is already weakened by mild function, and falls an easy prey to the overstress produced in traumatogenic function.

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STUDY NO. 5

EFFECTS OF TRAUMATOGENIC OCCLUSION IN THE PERIODONTAL TISSUES

STUDY NO. 5

EFFECTS OF TRAUMATOGENIC OCCLUSION IN THE PERIODONTAL TISSUESOutline - A. Gingival Changes

- (a) Experiments on Sheep (Box)
- (b) Experiments on Monkeys (Stones)

B. Changes in the Periodontal Membrane (Human)

- (a) Cementum fracture
- (b) Necrosis
- (c) Hemorrhage
- (d) Thrombosis
- (e) Collagenous Changes
- (f) Granulation Tissue
- (g) Fibrosis
- (h) Pericementitis fibrosa

C. Bibliography

EFFECTS OF TRAUMATOGENIC OCCLUSION IN THE PERIODONTAL TISSUES

A. Gingival Changes (a) Experiments on Sheep - (Box)¹
(b) Experiments on Monkeys - (Stones)²

(a) In the sheep the upper incisors and canines have been replaced by a dense, horny pad which covers the forepart of the upper jaw. The lower incisors and canines are arranged in a semi-circle and are adapted to the upper pad in browsing and grazing.

It is a matter of common observation that when a sheep feeds, the grass appears to be torn off rather than bitten off. This is accomplished by an abrupt sideways movement of the head after the grass has been pressed tightly between the lower front teeth and the pad, and is greatly facilitated when the outside edges of the teeth are sharp.

In some sheep the lower incisors and canines strike the pad only on a lingual plane on the crowns producing, through wear, teeth that are chisel-shaped and also extremely sharp on the labial edges. In others, on closure of the jaws the pad has a decided overjet and in these cases the pressure on the teeth is mostly axial, the tops of the crowns presenting flat surfaces. The observations of the writer, based on actual mobility tests, showed that, in general, in different sheep of approximately the same age, the lower anterior teeth under axial loading were tighter than those under oblique loading. And where the two classes of loading were manifested in individual sheep the teeth obliquely loaded that is to say under leverage, were markedly looser than the ones axially loaded.

On account of the peculiar features of the tooth-pad relationship in sheep, adapted for plucking herbage, it was decided to utilize this mechanism in experiment. Different procedures were carried out on several sheep, in which the loading on certain teeth was increased. This was achieved either by grinding down certain teeth so that others first received the thrust of the pad, or by increasing the height and thickness of the crown of the tooth through the insertion of a gold cap.

One experiment was conducted as follows: A gold cap was cemented on the crown of the lower right second incisor. It increased the crown height and thickness lingually in each case approximately one and a half millimeters, so that on closure of the lower anterior teeth against the pad, the brunt was first imparted to the capped tooth. This operation was completed on July 11, 1932. It was noted that this sheep on being released began at once to graze and from then on, with the exception of brief periods when it was subjected to examination, it lived as a member of the flock in its normal environment. The right second incisor exposed to abnormal functional conditions and the other anterior teeth were kept under fairly close observation until October 27th of the same year, when the sheep was killed.

After decapitation a 10% solution of formalin was immediately pumped through the carotid arteries and the head placed in a jar of the same solution. Following the regular processes of decalcification and embedding, the lower anterior teeth with their associated periodontal tissues were sectioned, mounted and stained.

Changes in the Lingual Crevice Depth. Using, in each case, sections cut axially through the pulp region, microscopic measurements of the crevices on the lingual of the four incisors, left and right first and second, were obtained. An eyepiece micrometer scale calibrated by means of a stage micrometer was employed. In the following table the depth of each crevice in millimeters is shown, together with the average measurement in each group:

DEPTH OF LINGUAL GINGIVAL CREVICES

	Left 2nd Incisor	Left 1st Incisor	Right 1st Incisor	Right 2nd Incisor (Capped tooth)
1.	2.907 mm.	2.850 mm.	2.907 mm.	3.705 mm.
2.	2.907 "	2.907 "	2.850 "	3.648 "
3.	2.907 "	2.793 "	2.793 "	3.705 "
4.	2.907 "	2.850 "	2.850 "	3.762 "
5.	2.850 "	2.850 "	2.850 "	3.705 "
6.	2.907 "	2.850 "	2.793 "	3.705 "
7.	2.850 "	2.850 "	2.964 "	3.705 "
8.	2.907 "	2.850 "	2.907 "	3.762 "
9.	2.907 "	2.850 "	2.850 "	3.762 "
10.	2.907 "	2.793 "	2.850 "	3.762 "
Average	2.895mm.	2.844mm.	2.861mm.	3.722mm.
Average of 3 control incisors				2.867mm.

It will be noted from the above figures that the average depth of the lingual crevice of the right second incisor is considerably greater than the average crevice depth of the other three teeth taken individually. The table shows an increase in the average crevice depth of the capped right second incisor, of 0.855 mm. over the average crevice depth of the three control incisors.

Preliminary determination of the depth of these crevices by probe-measurement was not attempted owing to possible infectious complications from trauma. And it should be pointed out that the edge of the gold cap did not extend into the gingival crevice nor did it encroach on the gingival margin.

It was observed that the amount of calculus in the lingual crevice of the right second incisor was much greater than that occurring in each of the control crevices.

(b) Experiments on monkeys (Stones)

The experiments of Stones were conducted on monkeys to determine the effects of traumatogenic occlusion on the gingival crevice as manifested by changes in the epithelial attachment and the underlying connective tissue. According to this investigator, in the untreated

monkey, the normal gingival crevice exhibits a lining epithelium which appears as a smooth band and unites with the tooth in a pointed manner, or as a blunt end joint. In other words, the junction is not distinguished by a downgrowth of epithelium along the cementum. Very few inflammatory cells are to be noted in the connective tissue underlying the epithelium.

From numerous histological investigations by different workers,^{3,4,5,6} it has been shown that early periodontal disease in man is characterized by a proliferation of the attachment epithelium along the cementum. With severance of the principal fibres from the cementum, the epithelium of the attachment has proliferated apically to cover the detached connective tissues, and at the same time maintaining organic connection with the cementum. Papillary processes also extend from the crevice epithelium into the underlying connective tissue. This connective tissue presents, as a rule, a marked infiltration of lymphocytes and plasma cells. As the disease process advances, separation of the epithelium from the cementum takes place with consequent deepening of the crevice. Stones⁷ has observed these early changes in the gingival tissues of dogs. As a result of these various observations, it was decided by him to regard these changes as criteria of early periodontal disease in an examination of the gingival tissues of the experimental monkeys.

In these experiments the production of traumatogenic occlusion was achieved by inserting raised fillings in three adjacent posterior teeth, one tooth being made higher than the others. This was done in six cases, in some animals the fillings being inserted in the maxillary teeth, in others, in the mandibular ones. In one monkey, the only tooth subjected to increased biting pressure was the left upper central incisor. On this tooth, a raised crown was placed. Taking into account the opposing teeth, in all, thirty-nine teeth were subjected to traumatogenic occlusion.

The experiments were begun in 1932, and were terminated at different times varying from ten to forty-three weeks. The treated teeth and their antagonists, and the associated periodontal structures, as well as the controls on the opposite side of the mouth, were sectioned serially. The effects of the traumatogenic occlusion, artificially produced, were determined by microscopic examination of the sections.

According to the abstract of the paper of Professor Stones, the results were as follows:

- "(a) Seven monkeys were treated.
- (b) In three monkeys, very definite changes analogous to periodontal disease were produced.
- (c) In three monkeys, less extreme changes were seen.
- (d) In one monkey there was no change.
- (e) In each animal, the pathological changes were usually observed in only the one tooth which took the greatest stress of the three that were filled, and the opposing one with which it articulated.
- (f) Of the 39 teeth subjected to trauma, eleven showed pathological changes in the subgingival tissues."

Stones points out that in a consideration of the practical implications of these investigations, the longest experiment was only

forty-three weeks. Definite changes were produced in thirteen weeks. Obviously the time factor in man may be greatly increased, at least to ten or twentyfold, and it is plain that the longer the tissues are exposed to the influence of traumatogenic function, the greater is the possibility of traumatic changes occurring in them.

In conclusion, it was pointed out, based on experimental and clinical findings, that traumatogenic occlusion is an etiological factor in the production of periodontal disease and in particular the type characterized by vertical pocket formation affecting only one or a varying number of teeth.

B. Changes in the Periodontal Membrane (Human)

(a) Cementum fracture⁸

Occasionally cases are found where fragments of cementum are torn away from the dentine. Such pieces are displaced into the periodontal membrane. These fractures usually occur in the periodontal zones of tension or pull zones. As a rule they tend to remain and, according to Kronfeld,⁹ the periodontal membrane assumes its former width over the fractured area.

(b) Necrosis

Necrotic areas, brought about by crushing, are frequently noted in histological examinations of the periodontal membrane. Wells¹⁰ has pointed out that "physical injury of even slight degree may bring on severe alterations in cells." The histological picture would indicate, in some instances, that the periodontal necrosis is not suddenly produced. The appearance of the border of the actual necrosis points to the possible concentration of calcium in this zone. Changes in the adjacent tissues point to an adaptation to the central changes and certain modified cells, cartilage-like in appearance, are to be noted.

As a result of necrosis of a given region in the periodontal membrane, no resorption of the alveolar bone can take place from this part. However, resorption of bone begins at points farther inwards, including adjacent supporting bone. Through resorptive processes originating in the marrow spaces, and on the outer border of the alveolar bone, an undermining process advances towards the necrotic region. When the bone bordering this region has been destroyed, the tooth is permitted to move.

(c) Hemorrhage

(d) Thrombosis

(e) Collagenous Changes

A collagenous change in regions of pressure in the periodontal membrane is frequently observed in microscopic specimens. The fibrillar outline can still be detected, with a few surviving nuclei. It is probable that this change is one that has been described as hyalinization by certain writers.

(f) Granulation tissue-

Orban¹¹ has shown in some of the push zones in the periodontal membrane of a tooth in traumatogenic occlusion, the presence of granulation tissue with resorption of cementum and alveolar bone. Other examples are occasionally seen in the push zones where the marrow tissue, following incorporation into the membrane, has suffered inflammation, the exudate cells and vascularity pointing to the formation of granulation tissue.

(g) Fibrosis-

In the push zones where the marrow spaces have been opened up through resorption of alveolar bone, and are found frequently incorporated in the membrane, the marrow tissue shows a definite fibrosis. This tissue assumes a new function when merged with the periodontal membrane, and associated with this is shown a new morphology assuming the qualities of the membrane. That this tissue is not a granulation tissue is shown by the absence of exudate cells and an atypical vascularity. Also the order of fibrosis is not like that following the healing of granulation tissue.

(h) Pericementitis Fibrosa-¹²

This lesion appears to localize itself most often in the periodontal tissues of teeth in traumatogenic function. It is a chronic and progressive lesion of the periodontal membrane which results in the rarefaction and destruction of alveolar bone. The process consists of the formation, in the pericementum, of a new tissue which on the one hand, changes the normal arrangement of the pericemental structures, and on the other, replaces the bone substance of the alveolar process. These changes in the periodontal membrane and the alveolar bone are concomitant lesions. This new tissue subsequently assumes the form of a fibrillar connective tissue.

In this new tissue, the reaction around the blood-vessels is a conspicuous feature. There is a proliferative change of the endothelial cells lining the blood-vessels leading, in many instances, to partial or complete occlusion of the vessel lumen. Endothelial cells are to be seen, also in the new tissue surrounding the blood-vessels, accompanied by occasional lymphocytes and plasma cells. A few fibroblasts are present. In other words, there is manifested a chronic and progressive tissue response wherein new fibroblasts make their appearance during the progress of a very low grade of chronic inflammation.

In the production of this reaction, however, there is no significant development of new capillaries, and the tissue cannot be looked upon as typical granulation tissue. Nevertheless, there has taken place a new growth of tissue, its various elements constituting at times a diffuse new structure, and, at other times, nodular and irregular masses of new tissue growth.

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STUDY NO. 6

THE PERIODONTAL POCKET

STUDY NO. 6

THE PERIODONTAL POCKETOutline - A. Sequence of Events in Pocket Development on the Cementum

- (a) Detachment of principal fibers
- (b) Epithelial proliferation
- (c) Separation of soft tissues from the cementum

B. The Periodontal Pocket

- (a) The Dental Wall
 - (1) cementum
 - (2) cementum cuticle
 - (3) calculus
- (b) The Periodontal Wall

C. The Periodontal Pocket and Absorption of Toxins

- (a) The investigations of Fish
- (b) The experiments of Graham

D. The Periodontal Pocket and Focal Infection

E. Bibliography

THE PERIODONTAL POCKET

A. Sequence of Events in Pocket Development on the Cementum

- (a) Detachment of the principal fibers from the cementum.
- (b) Epithelial proliferation to cover the detached connective tissues. Organic union with the tooth is at the same time maintained.
- (c) Separation of the tissues from the tooth surface.
 - 1. Before cuticle formation
 - 2. After cuticle formation

B. The Periodontal Pocket

(a) The Dental Wall

(1) Cementum¹

In man cementum is formed by membranous ossification. When calcification is commencing, a capsule-like investment is formed around the dentine-germ and enamel organ. This is known as the dental follicle. The follicle wall, as each portion of the dentine of the root is finished, is presented to it as a closely adherent vascular membrane which, on its inner or dentinal face, exhibits a layer of large cells known as cementoblasts. Through their agency the cementum is formed, the follicle wall opposite the root persisting as the pericementum or periodontal membrane.

Cementum is a hard tissue which forms the external covering of variable thickness over the roots of the teeth of man and many animals. Overlying the dentine, it begins at the amelo-cemental junction as a thin coverlet, and gradually increasing in thickness it extends to the apex of the tooth. The cementum is deposited in layers or lamellae which are thinner towards the neck of the tooth, and consist of a calcified matrix which is sometimes apparently structureless, at others finely granular or globular. Like bone, cementum contains Sharpey's fibers which appear as minute rods running through it at right angles to the lamellae. Through the medium of these fibers, the periodontal membrane is attached to the cementum. It is commonly thought that these fibers become calcified by a globular deposition of lime salts in them, as in bone.

Based on the presence of corpuscles or cells, cementum may be divided, as a rule, into two more or less distinct types. The first, the non-cellular, is represented in the cementum of the gingival third and usually part, if not all of the middle third of the root. Lacunae are practically constant in the apical third and frequently occur there in great numbers.

In a former paper,² the writer described the cellular cementum as follows: "Radiating from the lacunae in all directions are numerous fine channels which branch and subdivide into the cemental matrix. These are

known as canaliculi. They anastomose freely with those from adjoining lacunae and they also maintain a communication between the lacunae near the surface and the periodontal membrane. In the cemental lacunae, are found cells termed cement-corpuscles, fine projections from which extend into the canaliculi, bringing each cemental cell in close relation to a certain zone of matrix over which it has control. The cement-corpuscles communicate freely with one another by union of some of their fine protoplasmic extensions, the other offshoots extending into the canaliculi as delicate processes of variable length. The corpuscles in the lacunae near the surface of the cementum, by means of some of their extensions, seem to be joined to protoplasmic bodies in the periodontal membrane. There is, then, in cellular cementum a continuous network of living protoplasm throughout its matrix. The cement-corpuscles and their processes should be considered as being continually bathed in lymph plasma. This plasma circulates throughout the lacunae and canaliculi which form an inter-communicating network of lymph spaces similar to that found in bone. The nutrition of the cement-cells and the matrix is thus insured."

The majority of periodontal pockets are found upon the cementum of the gingival third, a non-cellular tissue. In other words, the cementum on the surface of which all pockets originate and over which they develop on their progress toward the apex is free from lacunae both near the surface as well as in its depths.

(2) Cementum cuticle

The cementum cuticle is a thin homogeneous keratinized layer formed by the crevicular epithelium during its proliferation along the cementum to cover the detached connective tissues. It holds for a time the cementum and the soft tissues in a state of organic union. With separation of the epithelium from the root this translucent pellicle remains, as a rule, firmly adherent to the tooth. It is continuous with that important component of Nasmyth's membrane, the secondary enamel cuticle of Gottlieb, and is its histologic counterpart. As it is formed in the same manner on the surface of the root as on the surface of the crown, Gottlieb has termed this layer in its entirety the "cuticula dentis." Weski confirms the existence of this dental cuticle, and has termed it the "epithelial cuticula"; and further, has divided it into a crown-part, the "enamel cuticula", and a root-part, the "cementum cuticula". The division into enamel and cementum portions, from a topographic standpoint, makes for clarity in periodontal nomenclature and teaching. It would appear, therefore, that this distinction, together with the fact that the term, "cuticula dentis", is commonly, though incorrectly used as a synonym for Nasmyth's membrane, presents apparent advantages in the use of an anatomic division of the dental cuticle.

Because of the relationship of the cementum cuticle with Nasmyth's membrane through the outer layer of the latter, and its histological and chemical similarity to it, a brief consideration of Nasmyth's membrane will be of interest at this point.

Alexander Nasmyth,³ a Scottish dental surgeon, first called attention to the existence of an exceedingly thin membrane which covers the enamel of the human tooth. Regarding this important structure great difference of opinion has been expressed. It has been described under various names, and is probably best known as "Nasmyth's membrane", or the "enamel cuticle".

Certain writers have used the term "cuticula dentis", conspicuous among whom is the name of Waldeyer, who described it only as an enamel covering. As he did not know that a cuticle was to be found on the cementum, this term did not accurately delineate his viewpoint.

Gottlieb's⁴ concept, briefly stated, is that first we must distinguish between a primary and secondary enamel cuticle, both together forming Nasmyth's membrane. After completion of the enamel, the ameloblasts form a membrane, the primary enamel cuticle about one micron in thickness, and chemically not different from other enamel. Then the ameloblasts, stratum intermedium, and stellate reticulum, disappear and before the eruption of the tooth the outer enamel-epithelium in the zone of eruption is transformed into stratified epithelium with cornification of the inner layers. Upon eruption, the stratified outer enamel epithelium unites with the oral epithelium and, as the tooth further erupts, this outer enamel epithelium separates from the cornified layer or secondary enamel cuticle. This secondary enamel cuticle is united with the primary enamel cuticle, and both together form Nasmyth's membrane, a cuticle about five microns in thickness.

Further, the base of the gingival crevice is located at any time at the deepest point of separation. The band around the enamel formed by the epithelial attachment first extends to the amelo-cemental junction, in other words, to the full extent of the outer enamel epithelium. When the base of the crevice arrives at the amelo-cemental junction, the epithelial attachment has already started downwards on the cementum. The layers of epithelium towards the root cornify in the same manner as the outer epithelium in the formation of the secondary enamel cuticle, and on separation, there remains on the tooth a cornified membrane.

James and Counsell⁵ have also called attention to the formation of this keratinized layer on the root-surface. They state: "As the condition advances, a proliferation of the epithelium along the cement is seen, a change which has been recognized by other workers. The cellular elements retain the characters of the subgingival epithelium. The very firm attachment to the cement by means of a newly-formed keratinized layer, continuous with that on the enamel, is a feature of much significance." Referring to the pocket they have stated further: "It is lined on the tooth aspect by the keratinized layer, cells attached undergoing atrophy, and on the gingival aspect by the main cellular part of the subgingival epithelium."

Lewis,⁶ too, has referred to this layer and has stated that its function appears to be that of binding down the epithelium to the cementum. He has written as follows: "Normally the free gingivae are attached to the cementum by means of epithelium, and in a large number of sections examined no hint is given as to the precise way in which the two tissues are cemented. Numbers of sections, however, clearly show the presence of an 'intermediary substance' which appears to be a highly resistant keratinized product of those epithelial cells which lie against the cemental surface. It consists of a hyaline layer lying between the cementum and the epithelium, which tends to become thinner as it approaches the end of the 'epithelial process' as this advances down the tooth-root. It clings very tenaciously to the cement wall, and is firmly attached to the epithelium which will itself tear rather than part from this substance."

The dental cuticle has long been known for its singular power of resistance to reagents, manifested chiefly by its insolubility in strong acids and alkalis. Some of its chemical qualities, according to Ebner, are as follows:

- (1) It is unchanged by soaking in water.
- (2) It does not dissolve in boiling water, strong acetic, hydrochloric, sulphuric and nitric acids.
- (3) It remains unchanged in caustic ammonia.
- (4) Boiled in caustic potash and soda it becomes white, somewhat loosened, but remains hanging together.
- (5) It burns with an ammonia odour and gives a calcareous spongy ash.

(3) Calculus

According to Prinz,⁷ all calcareous deposits about the teeth have one common source viz: saliva. From the standpoint of clinical differentiation, however, there is the division into supra-gingival and subgingival types. Supragingival calculus is localized on that part of the tooth surface which is crownward from the gingival margin. Subgingival calculus, on the other hand, is found on that portion of the tooth surface lying beneath the soft tissues, and accessible to saliva. And, as Kronfeld⁸ has pointed out, in a study of histologic sections, "the most apical extension of the mass of calculus found in any specimen of gingival crevice indicates the location of the bottom of this crevice." No concretions can be formed farther apically than the bottom of the crevice or pocket.

Composition of Calculus-

The following is the composition of dental calculus according to the analysis of Schecheetskey.⁹

Water and organic matter.....	22.07
Magnesium phosphate.....	1.07
Calcium phosphate.....	67.18
Calcium carbonate.....	8.13
Calcium fluoride.....	<u>1.55</u>
	100.00

Prinz¹⁰ gives us the following figures as the average of five analyses of salivary calculus.

Water.....	5.68
Water-soluble substances.....	5.24
Organic constituents (mucus and food debris).....	15.16
Magnesium phosphate.....	1.31
Calcium carbonate.....	9.14
Calcium phosphate.....	63.42

According to these findings, calcium and magnesium salts are the main crystalloids that tend to be precipitated from the saliva in calculus formation.

Formation of Calculus-

A survey of the many theories on calculus formation reveals the fact that there is, as yet, little agreement of opinion among investigators in this problem. According to Bibby,¹¹ the most common views fall into two main groups, viz:

- (1) that calcium salts are precipitated from the saliva by physicochemical processes, and
- (2) that this precipitation is brought about by bacteria.

In the first group, Kirk¹² attributed the precipitation to loss of CO₂ from the saliva. His stated opinion was as follows: "As saliva contains CO₂ in solution, the escape of this gas, which was the solvent, causes a precipitation of salts in the presence of the colloidal mucin in combination with which it deposits as tartar on teeth." Prinz believes that through the concentration of salivary colloids at the surface, the solvent power of the saliva is lowered and the calcium salts are liberated. The surface colloids form an insoluble pellicle which entangles the freed calcium salts and sinks into the medium to become attached to foreign surfaces. With successive films so attached, a concretion is formed.

Smith¹³ of Melbourne showed that desquamated epithelial cells liberate an enzyme, phosphatase, and according to Bibby, he believed that through the action of this enzyme on the organic phosphates of the saliva, inorganic salts are formed which enter into calculus formation.

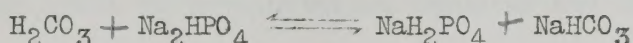
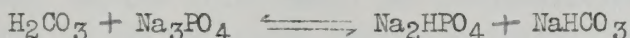
In the second group, viz: the viewpoint that the precipitation of calcium salts from the saliva is effected by microorganisms, Klebs and Goodrich and Mosley¹⁴ ascribed the cause to Leptothrix organisms. Soderlund¹⁵ held that the Actinomyces were responsible. Bulleid¹⁶ noted an intimate relation between calculus production and an organism he termed Leptothrix buccalis. Naeslund¹⁷ concluded from his investigations that both Leptothrix and Actinomyces were involved in calculus formation.

The extensive experiments of Bibby brought to light the important finding that, without the presence of microorganisms, no deposits of calculus were formed. Also, based on histological as well as bacteriological findings, Leptothrix organisms were found to be predominant. This investigator has pointed out that all the phenomena of calculus formation can not be explained on a bacterial basis. Certain physico-chemical processes may furnish a satisfactory solution for some of the facts. According to these experiments "the formation of calcareous deposits in saliva was prevented or retarded when samples were kept under an increased pressure of carbon dioxide." Bibby has stated in his summary: "It is suggested that physico-chemical processes lead to the initial precipitation of calcium salts from the saliva, while bacteria are important in fixing these precipitates on the teeth."

It is generally understood that the presence of CO₂ increases the solvent power of the saliva for calcium salts, and conversely, a loss of CO₂ lowers the solvent power and tends to precipitation. We know that the H-ion concentration varies as $\frac{H_2CO_3}{BHCO_3}$. If the CO₂ is

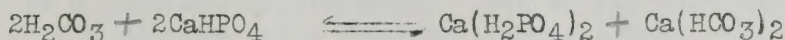
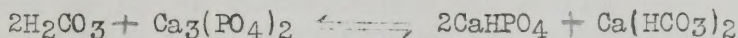
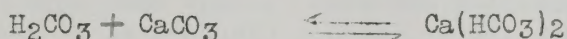
increased, then the ratio of H_2CO_3 to BHCO_3 is increased, therefore, the H-ion concentration is increased, or the pH is lowered. Conversely, if CO_2 is lost, then the ratio of H_2CO_3 is diminished, and therefore the H-ion concentration is decreased, or the pH is raised.

Professor Hunter* has suggested to the writer that the following reversible reactions might be helpful in an understanding of the changes involved in the precipitation of calcium carbonate and calcium phosphate in calculus formation:



In the body H_2CO_3 is everywhere present in such excess, that the first two of these reactions run completely from left to right, while the third reaches a point of equilibrium at which about one-fifth of the total phosphate has been converted into the mono-sodium salt. Under physiological conditions, therefore, neither Na_2CO_3 nor Na_3PO_4 is capable of existence, and only four of the substances represented in the equations (H_2CO_3 , NaHCO_3 , NaH_2PO_4 and Na_2HPO_4) are actual components of blood or tissues. Now, if in such a system the concentration of H_2CO_3 (or CO_2) is diminished, (as e.g. by free exposure to the air), all the reactions will be reversed. The mixture, as it loses CO_2 , will come to contain Na_2CO_3 and Na_3PO_4 . At the same time it will, of course, become more alkaline, i.e. its H-ion concentration will fall, or its pH will rise.

The behaviour of the corresponding calcium salts may be represented in an analogous manner, thus:



Within the fluids of the body, therefore, the only existent salts of calcium (with phosphoric or carbonic acids) are $\text{Ca}(\text{HCO}_3)_2$, CaHPO_4 and $\text{Ca}(\text{H}_2\text{PO}_4)_2$, all of which are at least moderately soluble. The saliva, as secreted, contains accordingly these three salts in solution, along with H_2CO_3 . In the oral cavity, CO_2 is given off, and the reactions represented in the equations are reversed. CaCO_3 and $\text{Ca}_3(\text{PO}_4)_2$ are formed, and, being highly insoluble, are thereupon precipitated.

Since CO_2 loss from the fluid in the pockets must be slight (Hall), Bibby has stated that physico-chemical factors, other than CO_2 loss, must be of greater importance in the precipitation of calcium salts in these regions.

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The presence of the following factors in gingival crevices and pockets, from a hypothetical standpoint at least, would seem to furnish a favourable basis for the formation of calculus on the tooth surface.

i. Calcium salts in a solution of colloids-

In the pocket both calcium salts are present in a solution also containing colloids. According to Wells¹⁸ "colloidal solutions hold in solution greater quantities of crystalloids than simple solutions for the reason that, at the surface of each colloidal particle, there is a zone in which the crystalloids are more concentrated than elsewhere, thus permitting more crystalloids to be dissolved in the solvent between the colloidal particles. On the other hand, the concentration of the crystalloids on the surface of the colloidal particles causes the colloids to serve as a starting point of precipitation whenever the crystalloids are in excess."

ii. Adherent vegetations of *Leptothrix* microorganisms on the tooth surface-

They serve, in some manner, to fix the precipitates. The great surface development of these bacteria would seem to provide an ideal holding medium.

iii. A rise in pH of the pocket solution-

It seems evident that a rise in pH would be accompanied by a precipitation of CaCO_3 and $\text{Ca}_3(\text{PO}_4)_2$ as previously described. The fluid in the great majority of gingival crevices and pockets is slightly alkaline in reaction, and in consequence, in many cases differs in this respect from the adjacent oral saliva. A rise in pH may be achieved, apart from CO_2 loss, through the growth and activity of a small microorganism known as *micrococcus gazogenes*.¹⁹

This microorganism was called by Lewkowicz, *micrococcus gazogenes alcalescens anaerobius*. It is a strict anaerobe, Gram negative and one of the smallest organisms known. From the evidence at hand, according to Professor Holman*, it is probably present in all mouths. This organism does not ferment any of the carbohydrates and similar compounds, forms no acid, and in all media, with or without sugar, progressively produces alkali. There is a possibility, therefore, that it may take part in the precipitation of the calcium salts from the pocket fluid.

(b) The Periodontal Wall²⁰

The wall of the pocket formed by the soft tissues is covered as a rule with epithelium to its deepest portion.

The gingival margin is created when, during the projection of the tip of the tooth into the oral cavity, in eruption, the outer enamel-epithelium joins with the oral epithelium. As Gottlieb has shown, with further eruption the epithelial attachment becomes separated from the enamel, and the base of the crevice or pocket is at any time denoted by the deepest point of separation. When the crevice-base has reached the

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enamel-line, the epithelial attachment has already advanced apically and is found upon the cementum. And when in pocket formation the base is to be found upon the cementum, the epithelium has further proliferated in advance along the cemental surface. In other words, with severance of the principal fibers from the cementum, the crevicular epithelium proliferates and extends apically to cover the detached connective tissues, at the same time maintaining organic connection with the cementum. When separation occurs, with or without cuticle formation, the pocket is created and, as stated previously, is lined to its deepest part on the soft tissue side with epithelium.

The periodontal pocket is comparable in many ways to an infected tonsillar crypt. The epithelial lining of the pocket is usually much thinner than that of the gingival mucosa. In certain zones actual small breaks in the epithelial continuity are to be noted. Both along the pocket-wall and at the base, through proliferation, long finger-like epithelial processes frequently extend into the underlying connective tissue.

The sub-epithelial connective tissue from the gingival margin to the pocket base, and for a considerable distance beyond, presents constantly a marked cellular infiltration, and in later stages chiefly of the plasma cell type. In mature cases, towards the gingival margin, remnants of the original fiber-bundles are to be noted, completely surrounded by dense masses of inflammatory cells. Lymphocytes, mononuclear wandering cells and young fibroblasts are also to be seen in these zones, which extend in projections, often wedge-shaped, between the fibrous tissue bundles above the crest of the alveolar bone. Hyaline droplets that have been developed in the cytoplasm of plasma cells are also to be seen persisting in the inflammatory zones. They are often called "Russell's fuchsin bodies". In the later stages the fibrous tissue mat has been reduced to a few parallel strands of fibers which separate enormous numbers of inflammatory cells. Extension of the cellular infiltration into medullary spaces of the alveolar bone is constant. It is noteworthy, on the other hand, that in the pericementum these marked cellular reactions are, as a rule, absent or slight except in advanced conditions. James and Counsell²¹ state that "a study of the extension of cellular infiltration provides little evidence in support of the view that lymphatic vessels from the corium pass into the periodontal membrane."

The presence of pus in the periodontal pocket is due mainly to the migration of polymorphonuclear leucocytes from the vessels in the soft tissues through the pocket wall to this locality. It is generally recognized that through the production of chemical substances by bacteria at the site of infection, the leucocytes are attracted to this region. At various positions along the pocket wall where extreme thinness of the lining epithelium, or actual breaks are present, many leucocytes are usually to be observed. Frequently actual ulceration of the surface is to be noted. According to Fish, the zone of infection is the debris of the pocket where streptococci exist in great numbers. These microorganisms are prevented from invading the normal tissues by the surface barrier presented by the polymorphonuclear leucocytes.

As a rule there is to be seen in the sub-epithelial connective tissue a rich capillary bed or network which is particularly conspicuous in those regions adjacent to masses of calculus upon the cementum. Occasionally small hemorrhages are noted as well.

C. The Periodontal Pocket and Absorption of Toxins

(a) The Investigations of Fish²²

These investigations conducted in the Meyerstein Laboratory for Dental Research, St. Mary's Hospital, London, have served to shed much new light on the problem of infection in periodontal disease. Through his brilliant researches, Fish showed, among other things, that the bacteria in periodontal infection, or so-called pyorrhea, are restricted, as to their presence, to the debris of the pocket.

It was found that in periodontal disease, the chronically inflamed investing tissues are always sterile. The marked round-cell infiltration, chiefly lymphocytes, is regarded as the typical reaction to toxic products formed in the pocket and which diffuse into environing living tissues. In this relation, Fish has stated: "The inflammation of the parodontal tissues could, therefore, only be due to toxic matter from the pocket soaking into the tissues at points where the pocket epithelium was ulcerated, and the infiltration of the tissues by round cells, which is the cardinal sign of chronic inflammation, is due to the predilection of lymphocytes for toxic material."

(b) The Experiments of Graham^{*23}

Graham conducted a number of experiments on animals in an endeavour to determine the effect on remote structures in the body of infection of sterile toxic material obtained from periodontal pockets. The material was procured fresh from periodontal pockets by the use of scalers, and placed in sterile Ringer's solution. When a certain concentration of pocket-detritus was achieved by this method, a sterile filtrate for injection was obtained by passing the septic solution through a Seitz filter. This procedure removed all bacteria. Starting with about 2 c.c. of sterile Ringer's solution, as a rule from 1/2 to 1 c.c. of sterile filtrate of the pocket contents was acquired for injection.

In the experiments, injection of the filtrate was undertaken in four groups of animals, twenty-five in all, viz: 4 cats, 14 guinea-pigs, 2 rabbits and 5 rats. Control animals living under conditions similar to those used for injection were killed and at times corresponding to the experimental animals. In a discussion of the findings of this investigation, Graham has pointed out that "the results obtained from this preliminary series of animals cannot be regarded as conclusive evidence of the precise effect of the filtrate from parodontal pockets." Nevertheless he has stated: "The experiment does however suggest that substances are elaborated in parodontal pockets which are highly toxic and tend to injure the liver and kidney of animals in the process of their elimination. It seems reasonable to conclude that in fifteen of the twenty-five animals, the toxic material proved fatal."

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D. The Periodontal Pocket and Focal Infection

A focus is an infected site; whether the bacteria leave that focus, either by the blood-stream or by the lymphatics, to reach distant sites of secondary infection determines whether the process of focal infection is going on or not. Billings has defined a focus of infection as a "circumscribed area of tissue infected with pathogenic microorganisms." Such infected foci should include only those in which a demonstrable zone of reaction is present and do not include the normal presence of bacteria in the superficial layers of the mucous membrane, between the epithelial cells and in the orifices of small mucous glands such as Bloomfield has suggested.

Holman²⁴ has defined the principle of focal infection as "the process of infection from an established ^{infected} focus and the secondary infection of other parts." He has stated further that "such an infected focus is not simply the site of the portal of entrance in bacterial invasion such as occurs in all infectious diseases, but is a local well-established infection capable of permitting the passage of its contained germs, from time to time, into the circulation."

The following factors interfere with the establishment of the process of focal infection:

- (1) An impermeable fibrous capsule;
- (2) A poor capillary response due to the relative inactivity of the bacteria in the focus.
- (3) The presence of high bactericidal substances in the blood-serum.
- (4) Perfect immobility of the part, which favours the healing process and lessens the opportunity for mechanical passage of bacteria into the blood-stream.

Recently Holman²⁵ has stated that "the factors determining the invasion of bacteria from an infected focus are largely dependent on the local circulation about and in the focus, and the relative quiescence or movement of the tissues. Dilatation of capillaries tends to facilitate such an invasion but sluggish circulation may permit the more invasive types of microbes, such as streptococci, to grow through or to produce local thrombi in the smaller vessels. The survival of these invading bacteria depends on their virulence (rate of growth) and on the inherent ability of the endothelial cells in a given organ to destroy them. The amount of damage resulting from virulent bacterial infections depends both on the pathogenicity of the parasite and on the resisting mechanism of the host."

The following are conditions which make a periodontal pocket a site for focal infection:

- (1) The periodontal pocket is an infected focus, i.e: it is an abnormal site of bacterial implantation with definite evidence of reaction on the part of the soft tissues. The thin epithelial layer or actual breaks in the epithelium, make the blood-vessels of the soft tissue wall of the pocket particularly liable to direct bacterial invasion from the pocket in traumatic conditions.

(2) Factors exist which favour the passage of bacteria into the blood-stream, viz:

- a) The presence of a rich capillary bed, with their thin walls evidenced clinically by the ease with which bleeding takes place under certain conditions, e.g: probing, scaling. This capillary bed renders the passage of bacteria into the blood-stream particularly simple.
- b) The presence of irritating agents, such as calculus, which produces a continued capillary response in the adjacent soft tissues.
- c) The mobility of the tooth in its alveolus, under occlusal stress as in mastication, swallowing and nervous habits, which interferes with the healing process. This is true of mobility for every infected region of the body. This factor of tooth-mobility is constant under normal occlusal conditions and greatly exaggerated in traumatogenic occlusion.

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